HEALTH EFFECTS ON INDIGINOUS POPULATIONS: A Review of the Literature

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AIR POLLUTION & HEALTH: HISTORICAL OVERVIEW

- Roman Empire: 80,000-100,000 metric tons of lead, 15,000 tons of copper, 10,000 tons of zinc, and >2 tons of mercury were used annually in industrial operations.
- 1300's: Silver and armor smithing banned in England due to contributions to air pollution.
- 1881: Chicago & Cincinnati regulate emissions of smoke and soot.
- 1895: Pittsburgh regulates emissions from steel mills.
- 1911: Boston notes air pollution has local, regional & national effects.
- 1930: Belgium's Meuse River valley: high concentrations of air pollutants held close to ground by thermal atmospheric inversion during a period of cold, damp weather: 60 deaths, most among elderly with history of heart and lung disease. Industry in the area included zinc smelting, sulfuric acid production, and glass factories.

Sand storm caused by wind erosion at Colby, Kansas, USA

AIR POLLUTION & HEALTH: HISTORICAL OVERVIEW

- 1935: U.S.A. dust storms of the great dust bowl (e.g. Kansas, etc).
- 1948: Donora, Pennsylvania, 20 people die due to exposures to air pollution from iron and steel mills, zinc smelters, and acid production. Cold, damp weather accompanied by a thermal atmospheric inversion.
- 1952: London, 4,000 people die from air pollution associated with coal burning during similar meterological conditions and fog between Dec 5^{th}-9^{th}. Most admitted to hospital were elderly or already seriously ill. Deaths correlated with concentrations of sulfur dioxide and smoke.

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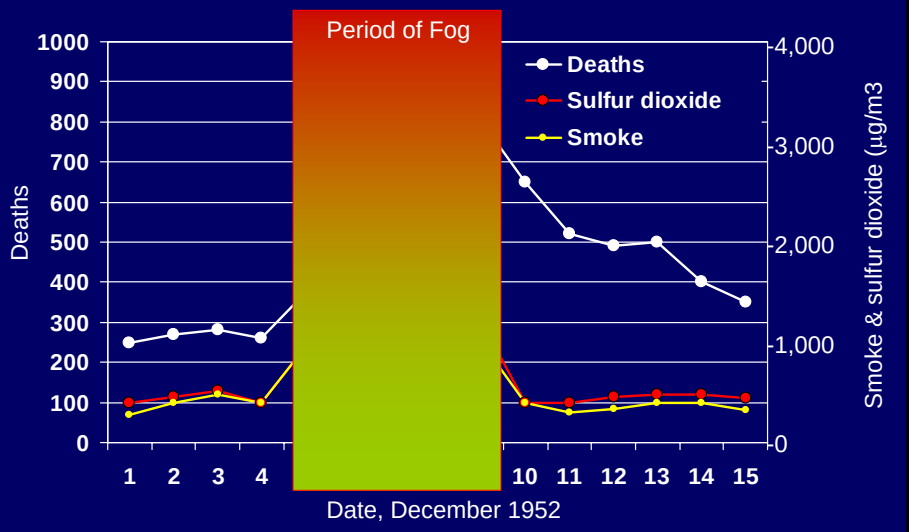
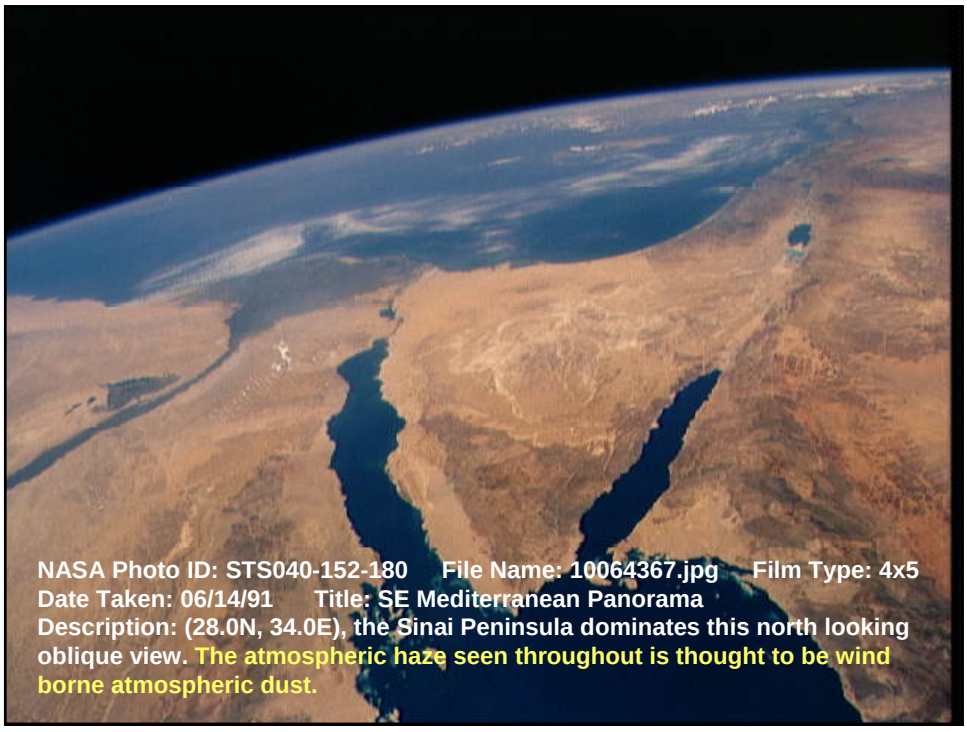
Daily Mean Pollution Concentrations and Number of Deaths During the London Fog Episode of 1952.



Source: Moeller, D.W. (2005). *Environmental Health.* Harvard Univ Press, Cambridge.



AIR POLLUTION & HEALTH: HISTORICAL OVERVIEW

1952: Policard & Collet report siliceous dust deposits without fibrogenic activity in the lungs of inhabitants of the Sahara desert.

1969: Fossati reports similar findings from the Libyan desert.

1974: Hirsch et al publish a report of simple siliceous pneumoconiosis of Beduoin in the Negev desert.

1983: Hawass describes a non-occupational dust pneumoconiosis ("desert lung") during the XVth International Congress on Radiology in Luxemburg. It is associated with micronodular dense shadows on routine chest radiographs in asymptomatic patients.

1986: Hawass & Nouh report pulmonary alveolar microlithiasis as a differential diagnosis for "desert lung". Patients may remain asymptomatic for years despite extensive radiological changes; attributed to inhalation of atmospheric sandy dust.

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Is the Desert Lung Syndrome (Nonoccupational Dust Pneumoconiosis) a Variant of Pulmonary Alveolar Microliththiasis? *Respiration 55:122-126.*
Mohamed S. Nouh (1989)

Four cases of pulmonary alveolar microlithiasis among patients with heavy exposure to sand particles for long periods. The author provides four case reports, 3 worked up for possible TB and the 4th as bilateral pneumonia. Two of the cases were biopsied. Very fine sand-like micronodulation of calcific density diffusely involving both lungs. Lung biopsy is not necessary to confirm diagnosis. Author hypothesizes that fine sand particles are responsible for triggering a hyperimmune response resulting in the formation of microlithiasis.

AIR POLLUTION & HEALTH: HISTORICAL OVERVIEW

- Air quality standards based on threshold models + safety margin.
- 1980's shifted to a size-specific standard using PM_{10}.
- 1990's emphasis placed upon $PM_{2.5}$; ***and***, documentation of association with increases in cardiorespiratory morbidity & mortality; growing appreciation that there may not be an exposure threshhold for $PM_{2.5}$.
- **1990-1993: Operation Desert Shield**

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http://www.henrysd.com/Photos/Ben_Hoffman/13-Sand%20Storm%20in%20Najaf.jpg

Al Eskan Disease: Desert Storm Pneumonitis

Military Medicine 157(9):452-462.

A.L. Korenyi-Both, A.C. Molnar, A.L. Korenyi-Both, & R. Fidelus-Gort. (1992).

Report of an acute desert-related disease attributed to the mixture of fine Saudi sand-dust and pigeon droppings which triggered a new clinicopathological entity, Al Eskan Disease. The composition of Saudi ambient sand-dust was studied and characterized. $PM_{1.0}$ (<1μm in diameter) present in substantial quantities in Saudi sand-dust and reported to be pathogenic causing hypersensitive allergic reactions (hyperergia*). The authors report that during the war, **~200,000 U.S. Army personnel reported on sick call & 22,743 were hospitalized; respiratory disease accounted for 43% of the sick calls.**

Hyperergia: A condition where the body has a much larger immune response to a given antigen than normal, including hypersensitive allergic reactions.

Respiratory Disease Among Military Personnel in Saudi Arabia during Operation Desert Shield.

Am J Public Health 83(9):1326-1329.

A.L. Richards, K.C. Hyams, D.M. Watts, et al. (1993)

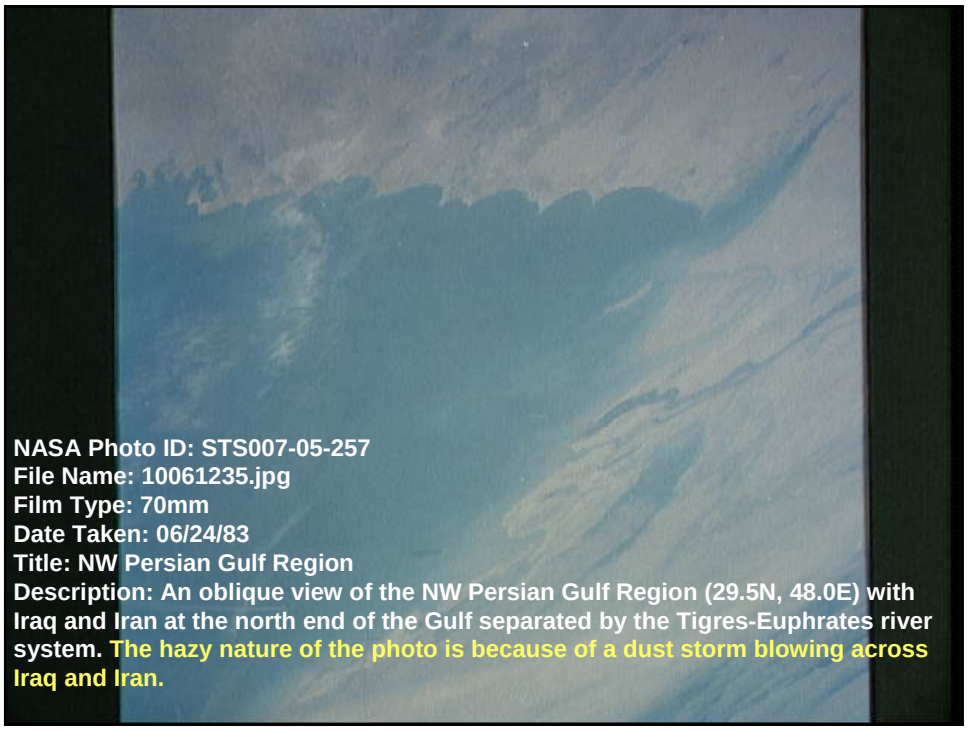
2,598 combat troops who had been stationed in NE Saudi Arabia for a mean of 102 days were surveyed. Symptoms reported were:

Sore throat = 34% Cough = 43% Chronic rhinorrhea = 15%

2% unable to perform routine duties due to upper resp symptoms.

Analysis by sleeping accommodations: complaints of sore throat & cough associated with sleeping in buildings with AC; complaints of rhinorrhea were associated with sleeping outdoor in tents.

Samples of surface sand from seven locations were analyzed by scanning EM & x-ray diffraction; most sand was respirable size PM_{10}.



NASA Photo ID: STS007-05-257
File Name: 10061235.jpg
Film Type: 70mm
Date Taken: 06/24/83
Title: NW Persian Gulf Region
Description: An oblique view of the NW Persian Gulf Region (29.5N, 48.0E) with Iraq and Iran at the north end of the Gulf separated by the Tigres-Euphrates river system. The hazy nature of the photo is because of a dust storm blowing across Iraq and Iran.

Sand encroachment towards a palmyard, in the south of Iran

ALLERGENS IN DESERT ENVIRONMENTS

Halonen et al (1997); Tuscon, AZ: skin tests of 895 children with asthma & allergic rhinitis for preferential associations and changes with age in children raised in a semiarid environment. Bermuda grass most prevalent among children with allergic rhinitis and controls; *Alternaria alternata* (mold) was most prevalent among asthmatics. Crude house dust and cat allergen reactions low in all groups. *Alternaria* was the only allergen independently associated with increased risk for asthma at both ages 6 and 11. Physician-diagnosed asthma prevalence at age-6 = 9.8%, at age-11 = 15.5%.

Ezeamuzie et al (1998); Kuwait, 505 blood donors: Kuwaiti nationals had a higher allergen sensitivity prevalence rate (50.2%) than did non-Kuwaitis (34.2%). Sensitization increased with age, but only among expatriates, as younger and older Kuwaitis were similarly sensitized. Polysensitization was common. Mite and plant pollens are major sensitizing allergens in this desert environment.

ALLERGENS IN DESERT ENVIRONMENTS

Ezeamuzi et al (2000); Kuwait: 810 patients with extrinsic asthma or allergic rhinitis (ages 2-76 years). Even in this desert environment, sensitization to molds is quite common among patients with allergic respiratory diseases (20.9% vs 5.8% among matched controls; asthmatics only, 45.8%). Mold could be an important factor determining asthma severity in this environment.

Shinn, E.A., Griffin, D.W., & Seba, D.B. (2003). Demonstrate that many species of fungal spores have characteristics suited to long-range atmospheric transport in clouds of desert dust. Hypothesize that atmospheric exposure to mold-carrying desert dust may affect human health directly through allergic induction of respiratory stress. Mold spores within these dust clouds may seed downwind ecosystems in both outdoor and indoor environments.

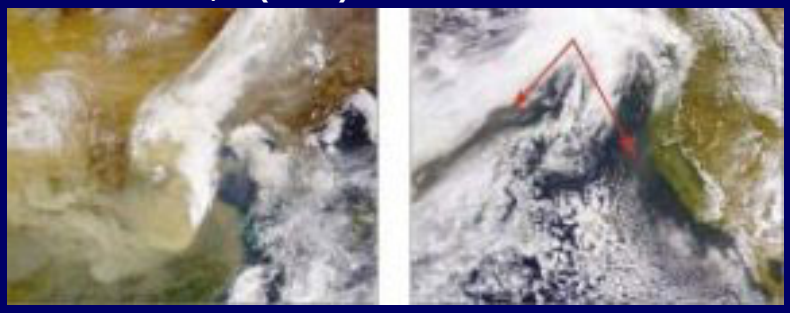
Dust Clouds Implicated in the Spread of Infection.
The Lancet 358:478.
McCarthy, Michael (2001).

"Griffin (Dale W.) says that now it has been established that micro-organisms can travel long distances by air, the next step is to determine whether enough of them can make the trip to cause disease in humans."

"Griffin and co-workers now plan to test African dust samples … to look for the presence of plant and livestock … (and) human pathogens, … They will also be examining hospital records … to see whether there in any correlation between asthma prevalence and African dust concentrations.

Sahara Desert Sand Storm, Mali, Africa
http://www.weru.ksu.edu/pics/nasa/10063608.jpg

Perkins, S.(2001). *Science News Online.*



On April 16, 1998, a strong storm system passing over the Gobi Desert sent a massive cloud of yellow dust southward into China (center of left image). The dust from a subsequent storm (arrows) crossed the Pacific and reached the western coast of North America on April 25 (right image).

Air Pollution and Daily Mortality in Seoul & Ulsan, Korea

Environmental Health Perspectives 107(2):149-154.

Lee, J.T., Shin, D., & Chung, Y. (1999).

The relationship between air pollution and daily mortality for the period 1991-1995 was examined. Daily death counts were regressed separately in the two cities, using Poisson regression on SO_2, O_3, and/or TSP controlling for variability in the weather and seasons. Independent pollution effects on daily mortality were observed even after controlling for either weather or seasonal variables. The study demonstrated increased mortality associated with air pollution at both SO_2 and O_3 levels below the current World Health Organization recommendations. **Particulates?**

Chinese Residents Battle Their Way Through a Sand Storm That Hit Zhengzhou

http://www.planetark.com/envpicstory.cfm/newsid/29929

Effects of the Asian Dust Events on Daily Mortality in Seoul, Korea. *Environmental Research Section A 90:1-5.* **Kwon, H.J., Cho, S.H., Chun, Y., Lagarde, F. & Pershagen, G. (2002).**

Asian dust clouds from the arid deserts of Mongolia and China impact the Korean peninsula in springtime. Industrialization of eastern China has increased the concern of Koreans as the arriving dust may contain Industrial pollutants. The authors studied the association between daily death counts and 28 dust days identified during the period 1995-1998. They used Poisson regression analysis adjusted for time trends, weather variables, and day-of-the-week. Using a 3-day moving average of exposure, non-accidental deaths from all causes increased by 1.7% (95%CI, -1.6 to 5.3); and, deaths of persons 65+y for cardiovascular and respiratory causes increased by 4.1% (95%CI -3.8 to 12.6).

Effects of the Asian Dust Storm Events on Daily Mortality in Taipei, Taiwan. *Environmental Research 95:151-155.* **Chen, Y.S., Sheen, P.C., Chen, E.R., Liu, Y.K., Wu, T.N., & Yang, C.Y. (2003).**

The authors studied the association between daily death counts and 39 dust episodes which served as index days during the period 1995-2000. For each index day, 2 symetrical comparison days were used (7d before and/after the index day). Effects were: respiratory-cause deaths increased by 7.7% 1-day after the index day; circulatory-cause deaths increased by 2.6% 2-days following the index day; and, total deaths increased by 4.9% 2-days after the index day. Air quality data used averages from 5 Taiwan EPA monitoring stations providing daily readings of SO_2, PM_{10}, NO_2, CO, & O_3. Temp/humidity also used.

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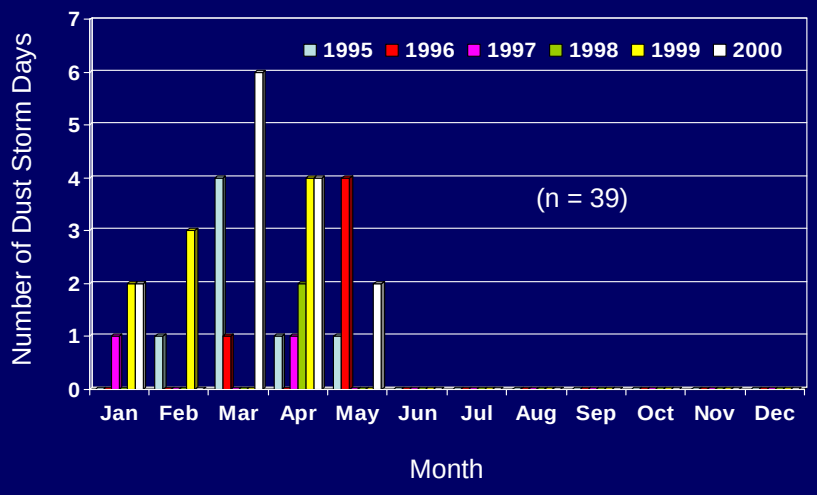
Asian Dust Storm Events (Days) in Taipei, Taiway, 1995-2000



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Mean Levels of Environmental Variables and Daily Death Counts: Dust Storm Days (Index Days) *vs* Comparison Days, Taipei, 1995-2000

Variable	Index Days (n=39)	Comparison Days (n=78)	*P*-value
PM_{10} (μg/m^3)*	**125.94 ± 33.61**	**57.80 ± 32.84**	**<0.0001**
SO_2 (ppb)	5.62 ± 3.67	5.07 ± 3.77	0.4542
NO_2 (ppb)	35.88 ± 10.74	34.97 ± 10.59	0.6631
CO (ppm*10)	11.72 ± 5.23	11.33 ± 5.18	0.7035
O_3 (ppb)	27.69 ± 9.40	20.91 ± 8.56	0.0002
Temp (C)	19.98 ± 4.03	20.45 ± 3.75	0.5375
Humidity (%)	68.64 ± 11.36	78.40 ± 9.28	<0.0001

Values are 24-hour averages. *PM_{10} in Asian dust storm events is composed of coarse crustal particles rather than combustion-related particles.

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Effects of Asian Dust Event Particles on Inflammation Markers in Peripheral Blood and Bronchoalveolar Lavage in Pulmonary Hypertensive Rats.

Environmental Research 95:71-76.

Lei, Y.C., Chan, C.C., Wang, P.Y., Lee, C.T., & Cheng, T.J. (2004).

Using a nose-inhalation system, 8 pulmonary hypertensive rats were exposed to concentrated ambient particles (CAPs) from an Asian dust storm that occurred 8-19 Mar 2002; 4 control rats exposed only to room air. 4 low exposure (315.6μg/m^3 for 6hrs), and 4 high exposure (684.5μg/m^3 for 4.5hrs). All animals sacrificed 36hrs after exposure and analyzed for markers of inflammation. Positive dose-response relationships between CAPs exposures and inflammation markers in BAL were demonstrated. (IL-6, ELISA, $p<0.001$)(peripheral WBCs in dose relationship; $p<0.001$)(Total cells in BAL increased, $p<0.05$; neutrophils increased, but not macrophage and lymphocytes)

What Properties of Particulate Matter are Responsible for Health Effects?

Inhalation Toxicology 12(Suppl 1):15-18.

B. Brunekreef (2000)

- Mass in Size: Respirable $PM_{2.5}$ *vs* "coarse mass" = [PM_{10} - $PM_{2.5}$].
- Number of Particles & Surface Area: <100-nm particles carry very little mass, but large surface area.
- Chemical Composition:
  - Transition metals and valence state.
  - Acidity.
  - Biogenic components such as pollen, bacterial endotoxin & mold and various allergens.

An Association between Air Pollution and Mortality in Six U.S. Cities. *N Engl J Med 329*(24):1753-1759.
Douglas W. Dockery, C. Arden Pope, Xiping Xu, John D. Spengler, James H. Ware, Martha E. Fay, et al (1993).

A prospective cohort study to est effects of air pollution on mortality, while controlling for individual risk factors. Survival analysis used data from a 14-to-16-year mortality follow-up of 8,111 adults in six U.S. cities. Mortality rates found strongly associated with cigarette smoking. After adjusting for smoking and other risk factors, significant associations were found between air pollution and mortality. The adjusted mortality-rate ratio for the most polluted of the cities as compared with the least polluted was 1.26 (95% CI, 1.08 to 1.47). Air pollution was positively associated with death from lung cancer and cardiopulmonary disease but not with death from other causes considered together. Mortality was most strongly associated with air pollution with fine particulates ($PM_{2.5}$), including sulfates.

Episodes of High Coarse Particle Concentrations Are Not Associated with Increased Mortality. *Environmental Health Perspectives 107*(5):339-342.
Joel Schwartz, Gary Norris, Tim Larson, Lianne Sheppard, Candis Claiborne, & Jane Koenig (1999).

$PM_{2.5}$, but not coarse particle concentration, was associated with increased mortality in 6 U.S. cities. Others argued that it could result from differences in measurement error between the two size ranges. Fine particles ($PM_{2.5}$) are primarily from fuel combustion, whereas coarse particles (i.e., particles >2.5µm and <10µm) are crustal material. One way to determine if coarse particles are a risk for mortality is to identify episodes of high concentrations of coarse, but not fine, particles. Spokane, Washington, is subject to occasional dust storms. Between 1989 & 1995, 17 dust storms occurred in Spokane.

1 of 4

Joel Schwartz et al (1999). *Environmental Health Perspectives 107*(5):339-342.

The 24-hour mean PM_{10} concentration during those storms was 263 $\mu g/m^3$. Using control dates that were the same day of the year in other years (but with no dust storm) and that had a mean PM_{10} concentration of 42 $\mu g/m^3$, comparisons were made of the rate of nonaccidental deaths on the episode days *versus* nonepisode days. The relative risk (RR) was found to be 1.00 [95% CI, 0.81-1.22] on the episode days.

Defining episode deaths as those occurring on the same or following day as the dust storm produced similar results (RR = 1.01; [95%CI, 0.87-1.17]).

Schwartz et al concluded that coarse particles from windblown dust are not associated with increased risk of mortality.

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***Environ Health Perspect* 108(1), January 2000**

Coarse Particles and Dust Storm Mortality

Schwartz et al restricted their analysis to mortality occurring on "the day of the dust storm or the following day." Therefore, they may have not observed any delayed rise in mortality that could have occurred later because of the "immediate" exacerbation of acute respiratory infections in highly susceptible individuals, such as those with preexisting COPD.

David T. Mage
U.S. Environmental Protection Agency
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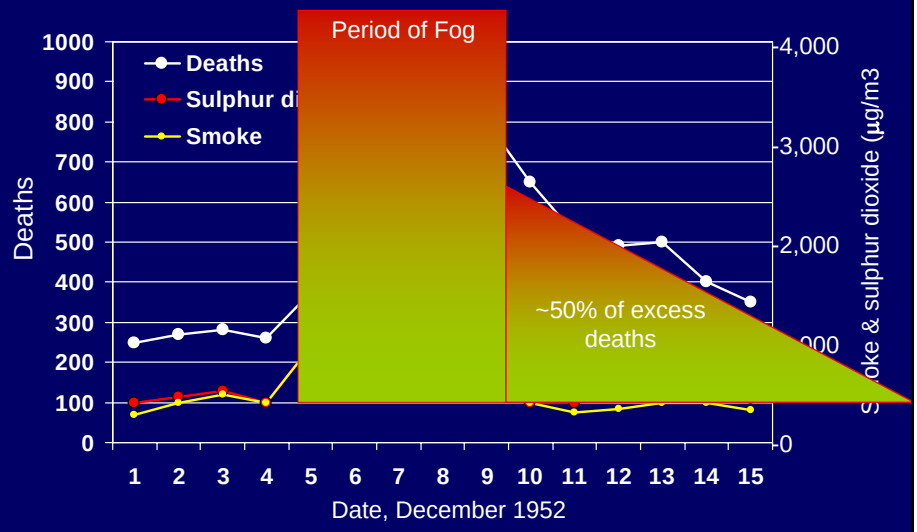
Daily Mean Pollution Concentrations and Number of Deaths During the London Fog Episode of 1952.



Source: Moeller, D.W. (2005). *Environmental Health.* Harvard Univ Press, Cambridge.

***Environmental Health Perspectives* Volume 108, Number 1, January 2000**

SCHWARTZ'S RESPONSE: Coarse Particles and Dust Storm Mortality

Dust Storms

In response to our paper reporting no excess risk of death in Spokane following dust storms when compared to appropriately chosen controls (*1*), Mage cites an article from the 1930s that anecdotally reports high rates of respiratory illness during the dust bowl period in the Midwest. This period, which coincided with the Great Depression, was a period of extreme social and economic stress. Since our paper was published, a reanalysis of three diary studies by Neas and Schwartz (*2*) has reported that lung function and lower respiratory symptoms were associated with fine particles ($PM_{2.5}$) but not coarse particles. Gold et al. (*3*) reported that heart rate variability is associated with fine particles ($PM_{2.5}$) but not coarse particles. These new results confirm our findings.

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Epidemiological evidence of effects of coarse airborne particles on health (2005). *Eur Respir J 26*:309-318

B. Brunekreef & B. Forsberg.

Institute for Risk Assessment Sciences, Utrecht Univ, The Netherlands.
Dept of Public Health & Clinical Medicine, Umea Univ, Umea, Sweden.

Studies on health effects of airborne PM have focused on PM_{10} or $PM_{2.5}$. The coarse fraction of PM_{10}, particles >2.5 µm, has only been studied recently. These particles have different sources and composition compared with $PM_{2.5}$. **Time series studies relating ambient PM to mortality provide the evidence for an independent effect of coarse PM on daily mortality**, but in urban areas evidence is stronger for fine particles. **In studies of COPD, asthma and respiratory admissions, coarse PM has a stronger or as strong short-term effect as fine PM, suggesting that coarse PM may lead to adverse responses in the lungs. There is also support for an association between coarse PM and cardiovascular admissions.**

Key Airborne Pollutants: The Impact on Health.
Science of the total Environment 334-335:9-13.
Maynard, Robert (2004)

- Time-series analyses reveal associations between air pollution and adverse health still exist; studies associate air pollution with hospital admissions & ER visit rates for treatment of cardio-respiratory diseases and with daily mortality rates.
- Prospective cohort studies (Dockery et al, 1993 [6 U.S. cities]; Pope et al, 1995) reveal shortening of life expectancy associated with inhabiting a relatively polluted city.
- **An independent group of Canadian experts (Krewski et al, 2000) estimated that 0.2-0.5 million life years could be gained per 1µg/m3 fall in $PM_{2.5}$ level.**

Ambient Particle Inhalation and the Cardiovascular System: Potential Mechanisms. *Environmental Health Perspectives 109*(suppl 4):523-527. **(A review article)**

K. Donaldson, V. Stone, A. Seaton, & W. MacNee (2001).

Air pollution episodes have led to deaths among persons with CVD and respiratory disease. The **ultrafine** component of particulate matter ($PM_{0.1}$) may mediate some of the adverse health effects for which there is toxicologic evidence to support this contention. These particles enhance calcium influx on contact with macrophages. Oxidative stress is anticipated at the particle surface; this can be augmented by oxidants generated by inflammatory leukocytes. Atheromatous plaques form in the coronary arteries and are major causes of morbidity and death associated epidemiologically with particulate air pollution. In populations exposed to air pollution episodes, blood viscosity, fibrinogen, and C-reactive protein (CRP – an index of inflammation) were higher.

Editorial

Potential Role of Ultrafine Particles in Associations between Airborne Particle Mass and Cardiovascular Health. *Environmental Health Perspectives 113(8).*

R.J. Delfino, C. Sioutas, & S. Malik (2005)

Epidemiologic time-series studies have shown consistent associations of cardiovascular hospital admissions and mortality with outdoor air pollution, particularly mass concentrations of $PM_{2.5}$ & PM_{10}. Panel studies with repeated measures have supported these results showing associations between PM and risk of cardiac ischemia and arrhythmias, increased blood pressure, decreased heart rate variability, and increased circulating markers of inflammation and thrombosis. The causal components driving the PM associations remain unidentified, but epidemiologic data provide indirect evidence that products of fossil fuel combustion are important.

1 of 3

Editorial

Delfino et al (2005). *Environ Health Perspectives 113(8).*

Ultrafine particles < 0.1 µm (UFPs) dominate particle number concentrations and surface area and are capable of carrying large concentrations of adsorbed or condensed toxic air pollutants. It is likely that redox-active components in UFPs from fossil fuel combustion reach cardiovascular target sites. High UFP exposures may lead to systemic inflammation through oxidative stress responses to reactive oxygen species and thereby promote the progression of atherosclerosis and precipitate acute cardiovascular responses ranging from increased blood pressure to myocardial infarction. The next steps in epidemiologic research are to identify more clearly the putative PM casual components and size fractions linked to their sources.

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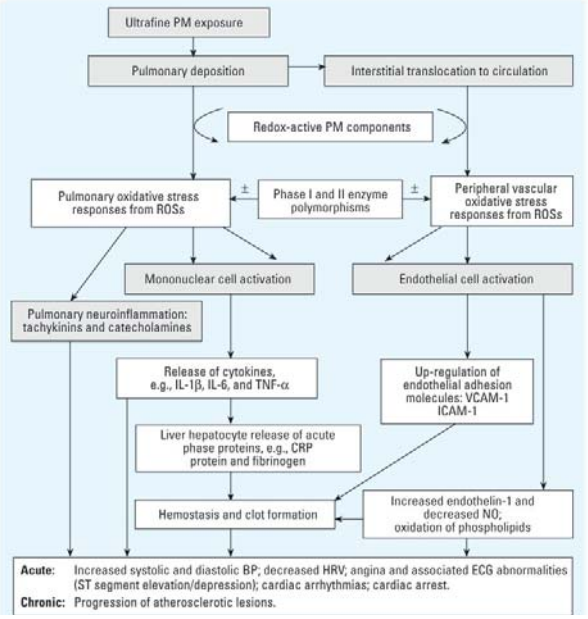
Potential Role of Ultrafine ($PM_{0.1}$) Particles in Associations between Airborne Particle Mass and Cardiovascular Health

Ralph Delfino
C Sioutas
Shaista Malik

Environ Health Perspectives 113(8): 947-955 (2005).



Hypothesized pathways to adverse cardiovascular health effects from UFP exposures. 3 of 3

***EHP*-in-Press**
Research

Fine Particulate Air Pollution and Mortality in Nine California Counties: Results from CALFINE

Bart Ostro
Rachel Broadwin
Shelley Green
Wen-Ying Feng
Michael Lipsett

Abstract

Ambient Air Pollution & Atherosclerosis in Los Angeles.

Environ Health Perspectives 113(2):201-206.

Nino Künzli, Michael Jerrett, Wendy J. Mack, Bernardo Beckerman, Laurie LaBree, Frank Gilliland, Duncan Thomas, John Peters, & Howard N. Hodis (2005).

Data on 798 participants from two clinical trials were used to investigate the association between atherosclerosis and long-term exposure to ambient $PM_{2.5}$. Baseline data included assessment of the carotid intima-media thickness (CIMT), a measure of subclinical atherosclerosis. Study subjects' residential areas were geocoded to assign annual mean concentrations of ambient $PM_{2.5}$. Exposure values were assigned from a $PM_{2.5}$ surface derived from a geostatistical model.

1 of 2

Kunzli et al (2005). *Environ Health Perspectives 113(2)*:201-206.

For a cross-sectional exposure contrast of 10 µg/m^3 $PM_{2.5}$, CIMT increased by 5.9% (95% confidence interval, 1-11%). Among older subjects (60 years of age), women, never smokers, and those reporting lipid-lowering treatment at baseline, the associations of $PM_{2.5}$ and CIMT were larger with the strongest associations in women 60 years of age (15.7%, 5.7-26.6%).

This is the first epidemiologic evidence of an association between atherosclerosis and ambient air pollution.

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WHO Ambient Air Quality Guidelines (as of 07 July 2004)

Compound	Guideline Value	Averaging Time
Ozone	120 micrograms/meter^3 (0.06 ppm)	8 hours
Nitrogen Oxide	200 micrograms/cubic meter^3 (0.11 ppm) 40 to 50 micrograms/meter^3 (0.021 to 0.025 ppm)	1 hour Annual
Sulfur Dioxide	500 micrograms/meter^3 (0.175 ppm) 125 micrograms/meter^3 (0.044 ppm) 50 micrograms/meter^3 (0.017 ppm)	0 min 24 hours
PARTICULATE MATTER	NO GUIDELINE VALUES SET FOR PMs BECAUSE THERE IS NO EVIDENT THRESHOLD FOR EFFECTS ON MORB/MORTALITY.	
Carbon Monoxide	100 milligrams/meter^3 (90ppm) 60 mg/meter^3 (50ppm) 30 mg/meter^3 (25ppm) 10 mg/meter^3 (10ppm)	15 minute 30 minute 1 hour 8 hours
Lead	0.5 to 1.0 micrograms/meter^3	Annual

Tallil Air Base, Iraq, on May 5, 2005. DoD photo by Staff Sgt. Darcie Ibidapo, U.S. Air Force. http://www.defenselink.mil/photos/May2005/050505-F-4903I-191.html

Thank You!
Bibliography is available.

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